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Letters to the Editor

Dear Editor

Kikuchi's disease and systemic lupus erythematosus

We read with interest the case report by Abba *et al.* (1) on Kikuchi's disease presenting as bilateral hilar lymphadenopathy. We feel the report fails to stress the importance of the association between histiocytic necrotizing lymphadenitis (HNL) and connective tissue diseases, in particular systemic lupus erythematosus (SLE).

We recently investigated a 29-year-old female with fever, cervical lymphadenopathy and a pruritic maculo-papular rash on the trunk and arms. The clinical picture was typical of other reports of Kikuchi's disease (2). Lymph node biopsy showed the 'pathognomic' features of Kikuchi's disease. The histological appearances were confirmed by independent review.

Over the following months, she developed a more florid rash with inflammatory plaques of discoid lupus, confirmed on skin biopsy. Her vision deteriorated in the left eye with retinal abnormalities consistent with SLE. Autoantibody screening confirmed the diagnosis of SLE with positive ANA and antibodies to double-stranded DNA. She has been treated by prednisolone and hydroxychloroquine, and is currently in remission.

The pathological feature of HNL in association with SLE has been reported in a small number of patients, and the SLE may be extremely florid in its activity (3,4). Our patients with SLE still required significant immunosuppressant treatment to suppress disease activity.

Like many reports on Kikuchi's disease, the report by Abba *et al.* continues to reinforce HNL as a brief benign self-limiting condition, and only mentions in passing the association with other systemic conditions. They have not excluded SLE or overlap syndrome in their patient, on the basis of the investigations reported.

It is essential that clinicians and pathologists appreciate that some patients with a pathological diagnosis of HNL may have severe, potentially life-threatening disorders. It may be best to avoid the term 'Kikuchi's disease', and simply describe the pathological feature of histiocytic necrotizing lymphadenitis.

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Reply to Drs Davies and Wathen

We appreciate the comments made by Drs Davies and Wathen on our report and agree with them fully that some patients presenting with Kikuchi's disease may indeed have potentially life-threatening disorders. This underlines the need for close follow-up of all patients with the histological diagnosis of histiocytic necrotizing lymphadenitis (HNL). As mentioned in our report, HNL and SLE share certain histopathological characteristics and indeed not less than 13 cases have been reported in the literature associating HNL and SLE (1). We have followed-up nine patients (of a group of 13 seen in our hospital) with histological diagnosis of HNL for at least 18 months. In all patients, lymphadenopathy and other symptoms subsided and no other features of connective tissue disease manifested. One of our patients with mixed connective tissue disease (MCTD) developed

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lymphadenopathy with histological feature of HNL. The development of HNL did not affect the course of her MCTD. The aim of our report was to highlight the peculiarity of presentation of this condition as bilateral hilar lymphadenopathy, which would be of immense interest to pulmonologists and pulmonary pathologists. Unlike their patient, ours had none of the American Rheumatism Association clinical criteria for the diagnosis of SLE (2), hence our failure to pursue laboratory criteria. We have followed this patient now for nearly 4 years. He remains well and asymptomatic.

The variable presentation and progression and the association with various physical, chemical, autoimmune and infective conditions indicates that this condition is a consequence of one or more (variable) stimuli. Until a unitary explanation is identified, it is best to treat each patient on merit.

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Dear Editor

Chylothorax in liver cirrhosis

We have read with great interest the article by Valdés *et al.* (1) in which two cases of chylothorax caused by liver cirrhosis were reported. The authors emphasize the rarity of chylothorax secondary to hepatic cirrhosis, which makes up approximately 1% of all chylothorax patients. We wish to report another patient with right cylothorax due to liver cirrhosis.

A 76-year-old woman was hospitalized in June 1988 because of dyspnoea of several days duration. Postnecrotic hepatic cirrhosis had been diagnosed 19 years before. In that time, portal hypertension was present. A splenorenal shunt and splenectomy were performed. Six months before admission, ascites developed. Paracentesis resulted in the removal of 3500 ml of milky liquid. Biochemical investigation of the peritoneal fluid was as follows: total protein 3·2 g dl^{-1}, lactate dehydrogenase (LDH) 87 IU l^{-1}, glucose 115 mg dl^{-1}, cholesterol 34 mg dl^{-1}, triglycerides 193 mg dl^{-1}, amylase 173 U l^{-1}, lysozyme 21·2 mg dl^{-1}, α_1-antitrypsin 125 mg dl^{-1}, carcino-embryonic antigen (CEA) 0·33 ng ml^{-1}, pH 7·40, PCO_2 43·5 mmHg, PO_2 73 mmHg, [HCO3-] 27·5 mEq l^{-1}, leucocytes 1100 mm^{-3}. Gram stain, acid-fast stain, culture and cytologic findings for malignancy were negative. On admission, clinical examination showed chronic hepatopathic stigmata, hepatomegaly, ascites and a right-sided pleural effusion confirmed by chest radiograph. Blood pressure was 120/70 mmHg, and heart rate was 84 beats min^{-1}. Blood analysis results were as follows: haemoglobin 12·8 g dl^{-1}, haematocrit 38%, leucocytes 7500 mm^{-3}, differential leucocyte counts normal, platelet count 256 000 mm^{-3}, prothrombin activity 53%, partial thromboplastin time 30·5 s, total protein 7·36 g dl^{-1}, total bilirubin 0·81 mg dl^{-1}, triglycerides 34 mg dl^{-1}, cholesterol 123 mg dl^{-1}, creatinine 2·24 mg dl^{-1}, blood ammonia 134 mg dl^{-1}, immunoglobulin G 3430 mg dl^{-1}, other biochemical parameters normal. Abdominal ultrasonography showed hepatomegaly without focal lesions and ascites. Thirteen thoracenteses, performed in a period of 3 months, resulted in the removal of 29 500 ml of milky liquid. Characteristics of the pleural fluid were as follows: protein 2·3 g dl^{-1}, pleural fluid: serum protein ratio 0·3, LDH 171 IU l^{-1}, pleural fluid: serum LDH ratio 0·4, glucose 90 mg dl^{-1}, cholesterol 46 mg dl^{-1}, triglycerides 121 mg dl^{-1}, pleural fluid: serum triglycerides ratio 2·2, amylase 195 U l^{-1}, lysozyme 22·2 mg dl^{-1}, α_1-antitrypsin 118 mg dl^{-1}, CEA 1·3 ng ml^{-1}, pH 7·48, PCO_2 45 mmHg, PO_2 48·3 mmHg, [HCO3-] 29·8 mEq l^{-1}, leucocytes 1200 mm^{-3} of which 60% lymphocytes, chylomicrons present. Gram stain, acid-fast stain, culture and cytologic findings for malignancy were negative. Total parenteral nutrition was started without reduction of